dle ear, both in acute catarrh and in purulent middle ear troubles. The acute catarrh infections have not been spoken of; they are rare, and are always due to a collateral hyperaemia and not to pressure of the exudate as has so often been thought. The symptoms are the same as in the ordinary first stage of Labyrinthitis. In purulent middle ear diseases which include the class of cases which the speaker has just covered so carefully, we may have a Labyrinthitis due to acute middle ear suppuration, always caused by collateral hyperaemia, whereas the chronic cases always infect the labyrinth through direct bony channels. No disease of the mucous membrane of the middle ear ever gives rise to a suppurative labyrinthitis. It is generally cholesteatoma, tuberculosis scarlatinal, etc. One per cent. of all chronic middle ear suppuration gives rise to a labyrinthitis (Hinsberg 1906). Seventy-five per cent. of post operative meningitis follows from a labyrinth infection not recognized or treated at the time of the operation (Zeroni 1905, 40 cases). There is 15 to 20% mortality from purulent labyrinthitis. In 68 cases of Hinsberg's, he lost none as the direct result of the operation. The labyrinth must be radically opened when we can diagnose a diffuse process, if we can discover a fistula or even if the horizontal canal shines as a dark mass through the bone. An exception must always be made if some hearing remain, for here the trouble has been encapsulated and a further operation is unnecessary. One side of the subject not spoken of is the infection of the labyrinth from the meninges. This is especially common in epidemic cerebro spinal meningitis where 10% of the survivors are absolutely deaf. A common source of infection of the labyrinth is through the blood, where the trouble is usually due to secondary syphilis, but whether it is here a labyrinthitis or a neuritis acustica is unknown. (b) Of the non-infective processes which lead to disease of the Labyrinth, I must mention first of all Otosklerose and secondary bleeding and emboli which occur in Leukaemia, pernicious anaemia purpura haemorrhagica, nephritis, etc.

Dr. C. F. Welty, San Francisco: This paper of Dr. Wintermute's covers so many different conditions that it is more or less difficult to make criticism. However, there are two points that I wish to speak of particularly. In the first place, a single semi-circular canal cannot be destroyed without destroying the entire chain. In healthy canals, the characteristic reaction can be produced by putting the patient in the proper position and applying the test. In the test of the patient in the revolving chair that the doctor referred to, he said he had a nystagmus to one side of 40", and to the other side of 20". This finding is in keeping with destroyed semi-circular canals—it does not make any difference if the patien's head is on the side or not. The 40" reaction is from the ampullary end and the 20" reaction is from the non-ampullary end—and in this case in question from the same canal. In the event of a good canal on this side, he would respond to the caloric reaction, which I believe is the best. In a differential diagnosis between serous and purulent labyrinthitis, the principal point of difference is that in serous labyrinthitis you do have rests of hearing while in the purulent you do not. In serous, no fever; in purulent, fever, and by the way, the fever is produced by the infection of the meninges, as the canals are not sufficiently large to contain pus enough to produce fever. Fever may also be produced by wound infection, intestinal intoxication and other existing maladies. They must be excluded before any importance can be attached to the findings. The differential point of diagnosis is of the utmost importance because on the one hand your patient may die if not operated, and on the other side a useful ear should not be destroyed. Again, your diagnosis may be so complicated that an absolute finding will be out of the question. In such instances I am inclined to the operative end rather than non-operative.

Dr. G. P. Wintermute, Oakland: Dr. Welty, in our talks, had told me about the matter of differentiating the serous from the suppurative cases by the rests of hearing and he gave Neuman as the authority. He is the best authority in the world and I am anxious to know more about it. The point he made is that you have arrests in hearing in serous cases without symptoms of meningeal irritation, headache and fever. You may have some fever, of course, from the middle ear, but I do not understand the reason for merely arrests of hearing in the serous cases. One other point Dr. Welty mentioned, in regard to the circumscribed process being limited to one canal, and I think the criticism is a good one, but in the case in which I tried it I depended upon turning tests alone. The horizontal turning nystagmus was one-half the duration to the affected side of what it was to the opposite, while the vertical and rottary nystagmus was of the same duration to both sides. I think it was a marked case. I was surprised when I got the result but the fact that the horizontal nystagmus reacted only one-half of that to the opposite side tended to show that the process was circumscribed to that horizontal canal.

(Concluded.)

THE EYE IN ITS SEMEIOLOGICAL ASPECT.*

By WM. F. BLAKE, M. D.

The field of investigation indicated in the title of this paper is a broad one and its full consideration would carry one so far afield that I am relieved that the presentation before this state meeting of two other papers, one on the relation of the eye to general medicine, the other on the surgical significance of papilloedema, will permit the restriction of this article to a much narrower compass.

I shall then confine myself to a consideration of eye palsies, corneal anesthesia, subjective changes in the field for form and color and early changes in the disc that are of help in the early diagnosis of intracranial growth and often of very definite aid to exact localization. I shall also briefly consider in passing implication of cranial nerves other than those strictly related to the eye when their involvement seemed a part of the clinical picture and in so far an aid to its interpretation.

Since new growths in certain parts of the brain are less productive of general localizing symptoms and of cranial nerve signs than in other more outspoken localities, I shall leave out of consideration these so-called silent areas, and consider only such subdivisions of the brain and brain stem as offer pathological signs coming particularly within the sphere of observation of the ophthalmologist and aurist. Closely correlated with any discussion of the pathology of the brain must be a reasonable familiarity with the laboratory and clinical studies of its anatomical divisions and of its functional localization. Any discussion of these problems is outside the province of this paper and is moreover unnecessary.

The comparatively recent investigation on blood pressure, cerebral circulation and cerebral localization of Sherrington and Hall, of Horsley, of Crile, and the publications of work from the Hunterian Laboratory at Johns Hopkins can be found in the files of the English and American Journals of

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Physiology, in Brain, in the Lancet and British Medical Journal, and in our best-known American publications. These journals are so accessible in private and in county medical libraries that even a brief summary of recent contributions to our knowledge of brain physiology and anatomy would here be superfluous.

You are familiar, too, with the physiological fact of the excretion of cerebro-spinal fluid by the ependymal cells lining the ventricles and covering the chorioid plexus and of its exit from the ventricle by way of foramina of Megendie and Luschka into the subarchnoid space, in the region of the roof and lateral recess of the fourth ventricle, also its subsequent escape into the sinuses by way of the pacchionian bodies and thence into the general circulation. Cushing has pointed out that there must be a means of escape of fluid directly into the veins of the head by minute openings, for in some of the lower vertebrates arachmoidean villae are not found. (These openings have, however, never been satisfactorily demonstrated.)

The presence, too, of the relatively large lakes of cerebro-spinal fluid in the basal cisternae and of the interpeduncula cistern in particular with its direct connection with the vaginal sheath of the optic nerve, will readily suggest to you the facility with which, under increase of intracranial pressure, fluid held under tension in the sheath of the nerve becomes the principal agent in the strangulation of the nerve head and the production of choked disc. The fact, too, that the brain is grossly divided into compartments by the stout dural membranes, the falx cerebri and the tentorium, will suggest itself to you as an explanation of the fact that a relatively large growth may develop in one part of the brain and remain for a long time without producing, by pressure, disturbances of motor or sensory of associated tracts in a distant lobe.

Leaving aside these considerations of the gross anatomy of the brain, we will briefly consider some ocular signs supposedly characteristic of and an aid to the definite localization of certain cerebral lesions.

Since an intracranial growth of a hard, or an infiltrating and destructive character, may long exist in a silent part of the brain without causing symptoms permitting of definite diagnosis, I have thought it best to consider those parts of the brain where lesions give early and definite eye symptoms. I shall then leave out of consideration tumors of the parietal and of the temporal lobe, of the motor areas, of the frontal lobe and of the optic thalmus. Even in these localities there is much that is of interest to us as oculists and aurists, when we recall the incidence of mind blindness and sensory aphasia in lesions of the parietal and temporal lobes, of motor aphasia in lesion of the motor area, disturbances of speech mechanism when the posterior part of frontal lobe is affected, and nystagmus and perhaps hemianopsia when the optic thalmus is the site of tumor. There is left then for more detailed consideration tumors of the occipital lobe, of the corpora quadringemina and cerebral peduncles, of the cerebellum, of the pituitary body and pons and medulla.

In tumors of the occipital lobe, in addition to the general signs of tumor, as headache, dizziness, nausea and prostration, we have these frequent though unfortunately not pathognomonic signs which are early developing hemianopsia, half blindness for colors, hemiachromatopsia, sensations of light and visual hallucinations in blind fields and in addition the absence of Wernicke's hemianopic pupillary sign. I have said these signs are not pathognomonic, for published cases show that hemianopsia may follow a lesion of the optic tract anywhere from the chiasm to the geniculate body and from there through the optic radiations to the visual cortex. Optic aphasia is frequent in tumor of this locality, and is perhaps explained by injury from pressure upon or infiltration of the association tracts between the occipital lobe and the first temporal convolution.

In tumors of the cerebellum there are in addition to the general signs of tumor an almost constant association of choked disc of high degree and of very rapid onset. As suggested by Paton, this may be due to the position of the tumor and the frequency with which formaenial openings in the roof of the fourth ventricle are occluded or the general lumen of this ventricle is obliterated by pressure, direct or transmitted. Headache is usually severe, constant and accompanied by vertigo and vomiting of severe type. The cerebellar attitude with ear on the side of lesion to the corresponding shoulder, the chin pointing away from side of lesion, is considered a sign of slight significance by many neurologists, while others credit it with considerable value. The gait is that of an intoxicated person. The ataxia in contradistinction to that of tabes is not increased by closing the eyes. It is not due to faulty sensory impressions from the outside, but is some defect in the central regulating mechanism of co-ordination. Ataxia is more marked in the upper than the lower limbs and usually confined to the homolateral side.

Additional eye symptoms are, first, nystagmus, which is characterized by being most marked when the patient looks to the side of lesion, in contradistinction to pure labyrinthine nystagmus; by being rather coarse and slow in movement and is often more marked in homolateral eye. Second, abduceus weakness; sixth nerve implication is very common in intracranial growths in widely distant parts of the brain (its great length making it particularly exposed to the influence of pressure); however, it is a very common symptom in tumors of cerebellum.

There is frequently present, too, a dissociated paresis of the opposite internal rectus so that while the eyes may strongly converge, associated later movement to side of the involved sixth is poor and from this dissociation of movement there frequently results another symptom of importance, a secondary conjugate deviation of the eyes away from side of lesion. These signs, I take it, could be caused by growth in or pressure on the associated tracts (Charts) in the peduncles of the cerebellum or by transmitted pressure to the corpora Quadrigemina and so indirectly to the nuclei of the third and fourth nerves lying

beneath these structures, in an instance where the tumor is situated far forward in the vermis.

In addition to these signs we have the particularly intense and rapidly oncoming choked disc, which is so rapid in onset and so intense in character that Mr. Marcus Gunn considers it strongly indicative of cerebella localization, even in the absence of other definite signs. What I have said of tumors of the cerebellum may give the impression that they are easy of localization, whereas the contrary is most true. It is one thing to make a diagnosis of a growth within the mass of the cerebellum, it is quite another thing to definitely locate it on one or the other side. It is to be hoped that the investigations of Barany of the phenomena of nystagmus will be of substantial aid in cerebella localization. These, briefly, are as follows: (a) If a normal man has a rotary vestibula nystagmus to the right side, he falls with closed eyes and feet together to the left. If head is turned 90% to the right, he falls forward; if turned 90% to left, he falls backward. In cases of cerebellar lesion with nystagmus the direction of falling is not influenced by the position of the head, and if in such a case we produce an experimental nystagmus, the falling which accompanies the nystagmus does not follow the type found in a normal ear. (b) If we let a normal individual with closed eyes touch with his forefinger or great toe some object held in front of him, and then with his eyes still closed withdraw the finger or toe and again point, he will, as a rule, point correctly. In cases of a cerebellar tumor or any lesion that implicates the association tracts, the patient in pointing will err to the side of his lesion.

Where the tumor is upon the base of the brain, in the cerebellar pontile recess, its exact locality may be more readily diagnosticated. Tumors of the cerebellar pontile angle may, depending upon their position, give symptoms chiefly referable to the cranial nerves, to the cerebellar peduncles, or to compression of the pons. Since a discussion of all three conditions would carry me too far afield, I shall omit the last two conditions and speak briefly of the first location (Chart). A momentary consideration of the nerve trunks and nuclei lying near or coming from the region, and particularly the exposed condition of the fifth, sixth, seventh and eighth nerves, will account for far the most common and pronounced signs of tumor of this region. Beginning with the fifth nerve we find anesthesia of the face on side of tumor, and where this is so slight as to be uncertain we may perhaps get corneal anesthesia and absence of corneal reflex. Weakness of the sixth nerve on the same side will be shown, when slight, by a nystagmus when the eyes are turned toward the suspected side and when pressure paresis is more pronounced by an internal squint, the eye looking away from the side of the tumor.

Weakness of the seventh nerve will show itself in a smoothing out of the lines of the face, by a lessened power of closing the eyelids against resistance and by impairment of expression. Pressure on the cochlear division of the eighth nerve may cause tinnitus and partial or complete nerve deafness, while vertigo and incoordination will result from involvement of the vestibular branch.

The ninth and tenth and twelfth nerves more often escape, though there are many proved cases in the literature presenting symptoms of difficulty of swallowing and hoarseness of the voice, of vagal attacks and paralysis of one side of the tongue.

Tumors of corpora Quadrigemina. On account of anatomical structure ocular palsies are usually among the early manifestations of tumor in this part of the brain. The nuclei of the oculomotor and fourth nerves lie near the aqueduct of Sylvius, and therefore pressure upon these nuclei or direct implication by tumor growths results in paralysis of muscles of the eye on both sides. Disturbances of hearing may also be expected and may be explained by the implication of the posterior colliculus of the corpora quadrigemina.

Internal ophthalmophegia is not a common symptom and is probably explained by the supposed position of the center for the ciliary muscle and the sphincter of the iris in the part supplying the superior rectus and inferior oblique. Evidence is strong that paralysis of the associated lateral movements of the eyeball is indicative of a lesion of the posterior longitudinal bundle near the sixth nucleus. Speller believes, from clinical and pathological evidence of the study of five cases, that persistent palsy of associated upward or downward movement is indicative of lesion near the aqueduct of Sylvius in the corpora Quadrigemina, and paralysis of lateral associated movements is to be explained by involvement of both post longitudinal bundles. If we assume that the nuclei of the superior rectus and inferior oblique muscles are in posterior part of oculomotor nucleus, we can understand why marked impairment of upward movement is present when other ocular movements are perfect. Personally I have never had the opportunity of seeing a case of tumor in this locality, though I have in my care at present a patient with ptosis of the left lid dissociated upward and downward eye movement, together with a double internal ophthalmophegia. I believe this patient's symptoms are due to a small hemorrhage in the region of the fourth and third nuclei, though the lesion must extend somewhat more anteriorly than is usual in tumors of their region.

(To be concluded in January Journal.)

MINORITY REPORT ON CONTRACT PRACTICE.

Submitted by D. A. HODGHEAD, M. D., San Francisco.

To the Officers and Members of the State Medical Society:

The question of contract practice has never been fully discussed in the medical organizations, nor has it ever been fairly presented by the medical journals. I shall not impose upon you by endeavoring to present all of the arguments, pro and con, but in a condensed way to give some views in defense of contract practice.

This practice is engaged in by the Army, the Navy, and the Pension Bureau of the United States government; by all state governments in their care of the insane; by all county governments in their